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Fallout Radiation: Effects on the Skin

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12.1 Introduction

Until recently it has been assumed generally that injury to the skin from ionizing radiation was not a serious hazard associated with the detonation of nuclear devices. However, in 1954 the importance of this hazard became apparent when widespread radiation lesions of the skin developed in a large group of people accidentally exposed to fallout radiation in the Marshall Islands following the experimental detonation of a large nuclear device. In addition to exposure of some 239 Marshallese people and 28 Americans, there were 23 Japanese fishermen exposed on their fishing boat. The radiation effects and skin lesions in this latter group have been described by Koyama *et al.* and others. Prior to that time, a limited number of skin lesions on the backs of cattle (Bird; Paysinger *et al.*) and horses (Atomic Energy Commission Report) has been noted from fallout following experimental detonations. In addition, exposure of the hands of several individuals who had carelessly handled fission product samples from a detonation resulted in the development of severe lesions (Knowlton *et al.*). Other cases of beta lesions in human beings of accidental or experimental nature have been described by Robbins *et al.*; Crawford; Low-Beer; Wirth and Raper; Conard and Tessmer; Kepp; Griffith *et al.* and Kepp, Miller and Reich; Nodle; and Witten *et al.* Some of the rather numerous studies on the effects of beta radiation on animal skin are reported by Henshaw; Raper and Barnes; Snider and Raper; Lushbaugh; Moritz and Henriques; Paysinger *et al.*; Brues; Cloudman *et al.*; Glucksmann; Kharchenko and Venolurov; Koletsky *et al.*; Minisov; Passonneau and Hamilton; Shubik *et al.*, Ungar *et al.*, Davis and Alpen; Tabachnick; Tabachnick and Weiss; and Tessmer.

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The recent accident in the Marshall Islands affords the first example of large numbers of lesions of the skin in human beings from fallout. Studies of these lesions in the Marshallese and Americans exposed have been documented (Conard *et al.*) and will be referred to frequently in this chapter.

Lesions of the skin induced by fallout are primarily due to the beta radiation from the fission products adhering to the fallout material and are, therefore, frequently referred to as beta burns. So-called beta burns of the skin may also result from accidental exposure to, or contamination with, radioisotopes used in science and industry. The possibility of such accidents must be considered seriously in view of the increasingly widespread use of radioisotopes.

12.2 Fallout Situations Resulting in Skin Damage

With detonation of nuclear devices, serious radiation injury to the skin is only associated with fallout situations where the radioactive material is sufficiently concentrated. Such concentrations are most likely to occur with close-in fallout, i.e., fallout that occurs within several hundred miles of the detonation. It seems probable that the fallout will be visible if serious acute skin damage is to result; however, this cannot be stated with certainty. In the Marshall Island accident, the extent and severity of the skin lesions were directly correlated with the amount of visible fallout. On the most distant of the contaminated islands, some 200 miles from the site of detonation, the fallout was not visible and no beta lesions of the skin developed among the inhabitants, whereas inhabitants in islands where fallout was visible developed skin lesions.

The world-wide deposit of fallout which occurs slowly from the troposphere or stratosphere does not result in skin injury since in this situation the fallout material is greatly diluted and reduced in amount.

Damage to the skin such as that seen with beta radiation does not result from the immediate penetrating gamma or neutron radiation associated with detonation of nuclear devices since the dose of such radiations necessary to severely damage the skin results in early deaths from damage to the bone marrow and the deep organs. Thus the skin burns observed in the Japanese casualties from the Hiroshima and Nagasaki bombs were not the result of ionizing radiation and were caused chiefly by thermal radiation. Fallout radiation associated with these bursts was insignificant.

12.3 Characteristics of Fallout Material

The chemical and physical make-up of fallout will vary according to the type of terrain or soil over which the detonation occurs. All fallout is particulate in nature, but the size and other characteristics of the particles will depend to some extent on the physical and chemical properties of the soil.

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The fallout associated with the Castle detonation, March 1, 1954, was a white, powdery material largely composed of incinerated coral. Aside from the radioactive component, the calcium oxide of the material was in itself irritating to the skin due to its caustic nature. Moreover, it was probably partly dissolved in the perspiration on the skin, thus increasing its irritating action. This also may have enhanced the radiation to the skin by brining the radioactive materials in closer contact with the skin. The presence of irritating chemicals on the skin is known to enhance the radiation effect (MacKee, Cipollaro and Montgomery). Fallout produced from other types of soil, not predominantly coral, might vary considerably in chemical and physical make-up and ability to irritate the skin. Color and particle size would also vary. For instance, siliceous type soils would probably form much less irritating fallout.

The particulate nature of the material results in a spotty distribution of lesions on the body. The Marshallese claimed that the material adhered closely to the skin and was difficult to brush off. This was borne out by the difficulties encountered in decontaminating the skin of the exposed individuals.

12.4 Sources of Radiation from Fallout

Figure 1 is a rough diagrammatic sketch showing the relatively uniform distribution of fallout on the ground, buildings, trees and personnel. The penetrating gamma radiation which is represented by the wavy, shaded areas penetrates many yards in air before it is attenuated appreciably, while the beta radiation represented by the stippling is completely attenuated in several feet. Damage to the skin results largely from the beta component in the fallout. By far the greatest part of the dose is contributed by the beta radiation while relatively little gamma radiation is absorbed. Estimates of the beta to gamma ratio vary widely up to 150/1 depending on the exposure conditions. Alpha emitters are usually not present in fallout to any great extent and due to their very weak penetrating ability, they are not likely to add significantly to the skin damage.

The skin dose results from two sources of beta radiation: the fallout material in direct contact with the skin (contact source) contributes by far the largest part of the dose to the skin, and the material on the ground (beta bath source) contributes a much smaller amount. The fallout in contact with the skin will usually be spotty in distribution and due to the particulate nature will result in multiple point sources of radiation on the skin. Though radiation from these sources is largely from the skin surface, it is possible that some deeper radiation may result from percutaneous absorption as well as penetration into the dermal region via hair shafts, sebaceous and sweat glands. Some of the fission products are water soluble, and

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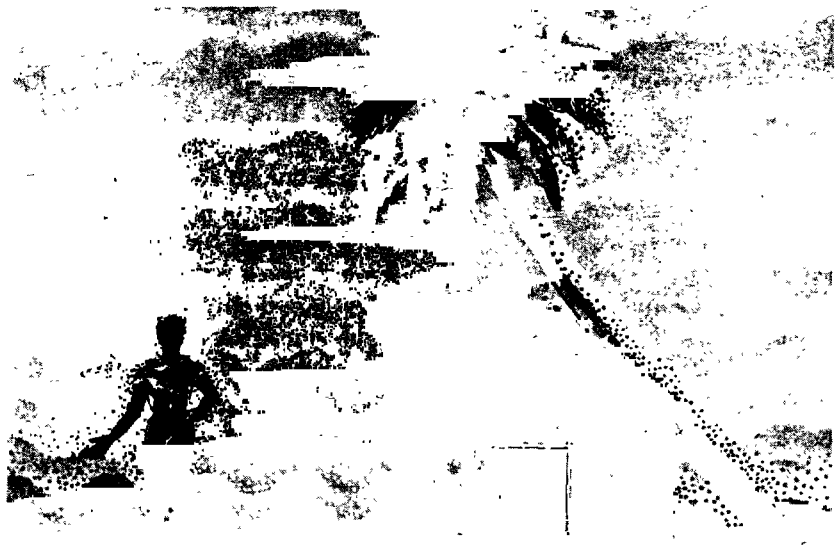

FIG. 1. Diagramatic sketch showing distribution of fallout. Gamma radiation represented by wavy shaded areas, beta radiation by stippling.

it is possible that some are lipid soluble, which would enhance this effect. Witten *et al.* have shown that thorium-x applied to the skin results in some percutaneous absorption and entry into the hair shafts and glands.

Beta dose to the skin from fallout on the ground will be largely confined to the lower parts of the body, particularly the feet and legs, since the beta particles are completely stopped in approximately 2 meters of air.

12.5 Estimation of Skin Dose

Measurement of beta doses to the skin from fallout is an exceedingly difficult problem due to the complicated spectrum of different energy beta emitters present, the non-uniform distribution on the skin, and the fact that practical dose meters have not yet been perfected which will adequately discriminate between the beta radiation and the contaminating gamma component.

The penetration of beta particles into the skin depends, of course, on the beta energies of the component isotopes. Each radioisotope has its own characteristic spectrum of beta energies up to a maximum energy. Relatively few particles are of the maximum energy, however, and the average energy (roughly one-third of the maximum energy) and the 50 per cent attenuation thickness of tissue are more meaningful in estimating skin effects. Thus an isotope emitting low energy radiation, confined largely to the dead, horny layer of skin, would be relatively ineffective; more energetic radiation, penetrating through the epidermis could result in transepidermal necrosis;

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and deeper penetration into the dermis could result in more severe ulcerating lesions. Figure 2 shows roughly the tissue depth necessary to produce 50 per cent attenuation of the beta particles from several isotopes.

In table 1 data from animal studies from several investigators show the

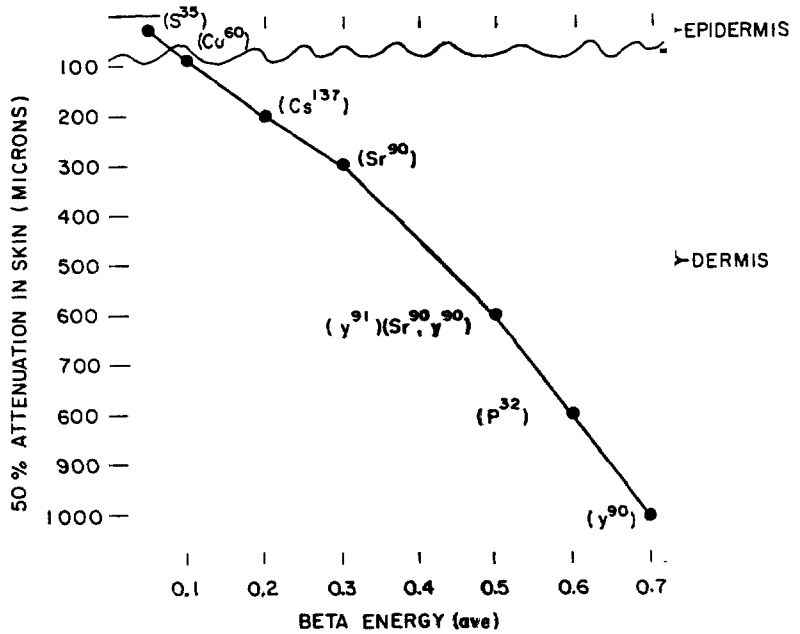

FIG. 2. Fifty per cent attenuation in skin, of various isotopes

TABLE 1

Surface Doses Required to Produce Recognizeable Epidermal Injury

INVESTIGATOR	ANIMAL	ISOTOPE	AVE. ENERGY	SURFACE DOSE
			mev	*rep*
Henshaw *et al.*	Rats	P^{32}	0.5	1,500–4000
Davis and Alpen	Rats	Sr^{90}	0.3	1,050
Davis and Alpen	Guinea pigs	Sr^{90}	0.3	2,500
Snider and Raper	Mice	P^{32}	0.5	2,500
Raper and Barnes	Rabbits	P^{32}	0.5	5,000
Lushbaugh	Sheep	S^{90}	0.3	2,500–5000
Moritz and Henriques	Pigs	S^{35}	0.05	20,000–30,000
Moritz and Henriques	Pigs	Ca^{60}	0.1	4,000–5,000
Moritz and Henriques	Pigs	Cs^{137}	0.2	2,000–3,000
Moritz and Henriques	Pigs	Sr^{90}	0.3	1,500–2,000
Moritz and Henriques	Pigs	Y^{91}	0.5	1,500–2,000
Moritz and Henriques	Pigs	Y^{90}	0.7	1,500–2,000

(Courtesy Brookhaven National Laboratory)

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energy dependence of beta particles from various isotopes in producing recognizable skin reactions. Note that the surface doses for threshold reaction (erythema, epidermal atrophy) are fairly dependent on the energy of the beta particles of the various isotopes. Thus it takes 20,000 to 30,000 rep from S^{35} (avg. energy 0.05 mev) to produce a reaction, while it takes only 1000 to 2000 rep of Sr^{90} or Y^{90} (avg. energy 0.3, 0.7 mev) to produce the same reaction.

Fractionation of the total beta dose given at intervals greatly reduces the skin reaction. For example, Jacobsen *et al.* have shown that in rats 200 rads of beta radiation (Sr^{90}-Y^{90}) given every 48 hours up to 12,000 rads total dose resulted only in epilation, while 3000 rads given in a single exposure produced wet desquamation in 14 days in all rats.

The degree of skin damage therefore is dependent on the absorbed dose at a certain critical depth in the skin. Moritz and Henriques found that the dose at 0.09 mm. depth of the pig skin (estimated to be the epidermal thickness) was constant within several hundred rep to produce transepidermal injury. Wilhelmy has also noted that it takes roughly the same dose of electrons and soft x-rays at the level of the subpapillary layer to produce erythema. On this basis, Parker has advocated the use of beta-detecting instruments with chamber walls corresponding in milligrams per square centimeter to the thickness of the relatively inert epidermal layer. Thus in expressing skin dosage, it is probably more informative to use the depth dose at a level corresponding to the basal cell layer of the epidermis.

Table 1 also indicates the species difference in skin sensitivity to beta radiation. Rabbits and sheep required larger doses than mice to produce the same effect with roughly the same energy beta. Porcine skin, which is reputedly more like human skin than other animals, apparently is more sensitive than the rabbit or sheep skin. Some of these differences, aside from species differences, may be due to variation in thickness of the epidermis of different species and differences in techniques used.

Table 2 shows beta dosage data from some human experiments and accidents found to produce various effects on the skin. These data must be interpreted with great caution due to differences in experimental techniques and dosimetry. The authors have taken the liberty of interpreting the severity of the skin reactions given by these investigators in degrees. A first degree reaction implies erythema and/or dry desquamation; a second degree, transepidermal necrosis with ulceration; and third degree, lesions which show deeper dermal involvement with breakdown and the development of chronic radiation dermatitis. It can be seen that there is a considerable variation in dose reported to produce the various reactions.

In the Marshallese the dose to the skin could not be calculated with any degree of accuracy due to the aforementioned reasons. The majority of the

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TABLE 2

Human Exposure to Beta Radiation

INVESTIGATOR	RADIATION	EST. DOSE	REACTION
		rep	
Wirth and Raper	P^{32}	635	1st degree (threshold)
Wirth and Raper	P^{32}	1180	2nd degree (threshold)
Low-Beer	P^{32}	143*	1st degree (threshold)
Low-Beer	P^{32}	7–17,000	2nd degree
Robbins *et al.*	Cathode rays (1200 kv)	1–2000	3rd degree
Knowlton *et al.*	Fission products (1 mev avg. energy)	3–4000	2nd degree
Knowlton *et al.*	Fission products (1 mev avg. energy)	5–10,000	3rd degree
Knowlton *et al.*	Fission products (1 mev avg. energy)	5–10,000	3rd degree
Knowlton *et al.*	Fission products (1 mev avg. energy)	8–16,000	3rd degree

* Estimated dose in 1st mm. layer.

(Courtesy of Brookhaven National Laboratory)

beta radiation was of low energy (avg. 0.1 mev, Sondhaus *et al.*) and accounted for the fact that most of the lesions were superficial in nature. However, there was sufficient penetration of more energetic components at the level of the hair follicles to result in temporary epilation. Due to the rapid attenuation of beta particles in tissue, the skin surface dose may have been quite high. The contribution of beta radiation to the skin of the Marshallese from the ground has been estimated by Sondhaus *et al.* to have been about 2000 rep to the feet, 600 rep at hip level and 300 rep to the head. These doses were insufficient in themselves to produce detectable lesions, though they probably contributed significantly to the severity of the foot lesions that occurred.

12.6 Effects of Fallout Radiation on the Skin

12.6.1 *Acute Effects.* In general beta radiation effects on the skin are similar to effects produced by more penetrating radiation such as gamma or x-radiation (Low-Beer; MacKee, Cipollaro and Montgomery; Warren; Nodl; and Walbach). However, since beta radiation is less penetrating it produces more superficial lesions with less damage to the dermis. The lesions are more like those produced by grenz-rays and ultraviolet rays (MacKee, Cipollaro and Montgomery; Ellinger). Consequently, they are usually less painful and heal more rapidly. The time sequence of beta lesions varies considerably with the dose to the skin. A general description of the sequence of changes is presented below.

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12.6.1.1 *Early effects.* During the first 24 to 48 hours after exposure, itching, burning, or tingling sensations of the skin are usually experienced. These symptoms may also involve the eyes with accompanying lachrymation. As pointed out earlier, fallout of an alkaline nature may contribute to this symptomatology. The above symptoms occurred in many of the Marshallese. In more severely damaged skin, erythema, edema and areas of blanching may be noted. Erythema was not observed in the Marshallese, perhaps due to the dark color of the skin.

12.6.1.2 *Latent period.* The early signs and symptoms usually disappear within a few days and a relatively asymptomatic latent period ensues. The length of this latent period may vary from a few days to several weeks and is related to the dose to the skin; the higher the dose, the shorter the latent period. In the Marshallese, the more heavily exposed group developed lesions about 2 weeks after exposure, a week earlier than the less heavily exposed groups.

12.6.1.3 *Development of gross lesions.* Following the latent period the evidence of skin damage becomes apparent with intensification of signs and symptoms. A secondary wave of erythema may be seen along with gross changes in the skin. Such changes may be in the form of simple tanning or more marked pigmentation with the formation of macules, papules, or raised plaques of thickened pigmented skin. Mild lesions may cause only slight itching and burning and superficial desquamation from the center of the lesion outward, leaving depigmented thinned areas of epidermis which gradually repigment and heal the following week or so. In the more heavily exposed Marshallese group of 64 people, about 90 per cent developed multiple, spotty, pigmented lesions on exposed parts of the body. Most of these lesions were superficial in nature (see figures 3, 4, and 5). More severe exposure to the skin results in vesiculation and ulceration. Such lesions may be quite painful and secondary infection may occur. They require longer to heal and may result in some degree of atrophy and scarring of the skin. Repigmentation may be long delayed or may never be complete. Only about 20 per cent of the Marshallese group referred to developed ulcerating lesions and secondary infection occurred in a few cases. Lesions on the dorsum of the feet were generally the most severe, showing bullae formation followed by ulceration (fig. 6). At 8 years after exposure some of these lesions continue to show incomplete repigmentation of the skin with atrophy or scarring in some cases (see fig. 7).

Epilation may occur along with the development of the skin lesions. The head region is more sensitive to epilation than the axillary, pubic, or eyebrow regions. If the radiation dose to the follicles has not been too high, regrowth of hair commences in several months. Permanent epilation may result if the skin dose is high. Usually by 5 or 6 months, regrowth of hair is

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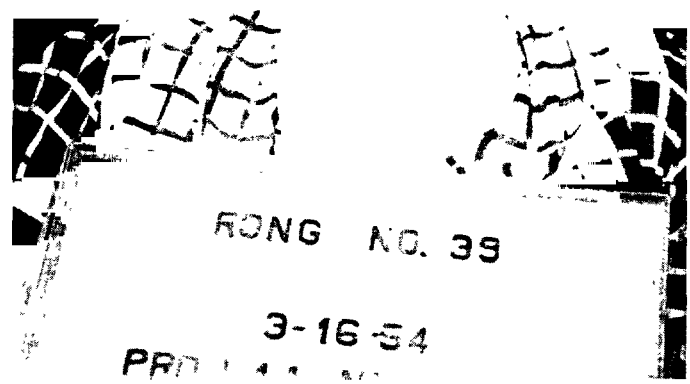

FIG. 3. Early hyperpigmented maculopapular neck lesions at 15 days. Case 39, age 15, F.

complete. In the Marshallese group, spotty epilation of varying degrees occurred in 90 per cent of the children and about 30 per cent of the adults (fig. 8). Regrowth of hair commenced in all cases about 3 months postexposure and by 6 months, hair was of normal color, texture and abundance (fig. 9). Though change of color of hair from black to gray has been frequently observed in animals (Hance and Murphy; Chase), regrowth is usually of normal color in the human being. However, Conard and Tessmer have reported a case in which regrowth of the hair of the eyebrows (previously black) regrew white in a lesion presumably due to fission product contamination.

Van Scott and Reinerton have reported that microscopic examination of plucked hairs from the scalp of individuals receiving epilating and subepilating doses of radiation reveals dysplastic changes in the bulbs of the hair roots.

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FIG. 4 FIG. 5

FIG. 4. Extensive lesions in 13-year-old boy at 46 days post exposure. Case 26.
FIG. 5. Same case as in figure 4, 6 months after exposure showing healed lesions and regrowth of hair.

Some biochemical changes have been reported in the skin of guinea pigs exposed to beta radiation (Tabachnick; Tabachnick and Weiss). Doses of 3000 rep (Sr^{90}-Y^{90}) resulted in early local increases of the acid soluble constituents (urocanic acid, ammonia and total free amino acids) and microscopic changes characteristic of second degree radiation burns. There was also progressive increase in epidermal RNase activity reaching a peak of three times control values by 3-weeks.

12.6.2 *Chronic Effects, Carcinogenesis.* Following large doses of beta radiation, imperfect healing may result. Damage to the vessels of the dermis may result in sufficient impairment of circulation to cause cycles of breakdown and repair of the epidermis or chronic, indolent ulcers may result.

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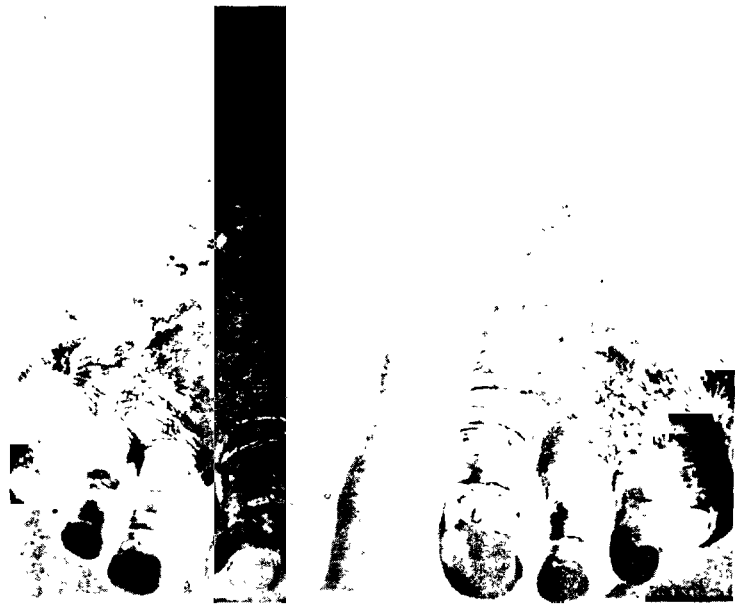

FIG. 6. Hyperpigmented raised plaques and bullae on dorsum of feet at 28 days after exposure: one lesion on left foot shows deeper involvement. Feet were painful at this time.

FIG. 7. Same case as in figure 6, 6 months later. Foot lesions have healed with repigmentation, except depigmented spots persist in small areas where deeper lesions were.

Also commonly seen are atrophy, scarring, keratosis and telangiectatic vessels. The hair follicles, sweat, and sebaceous glands may be injured sufficiently to result in permanent epilation and dryness of the skin. Such lesions are fertile ground for the later development of malignant change.

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FIG. 8

FIG. 9

FIG. 8. Epilation in 7-year-old girl at 28 days after exposure. Case 72.
FIG. 9. Same girl as in figure 8, 6 months after exposure showing complete regrowth of normal hair.

Lesions of the skin resulting from beta radiation are less likely to result in chronic radiation dermatitis than are the lesions produced by more penetrating radiation such as are sometimes seen following x-ray or radium therapy.

On the more recent surveys of the Marshallese (6 to 8 years postexposure) Conard *et al.* report that an increasing number of people show pigment changes occurring at the sites of previously healed burned areas (see figure 10). The majority of these changes were in the form of pigmented nevus-like lesions which appeared grossly benign. A biopsy of one of these lesions showed no evidence of malignant change.

Malignant changes in the skin have been reported in animals following beta irradiation (Raper and Barnes; Brues; Glucksmann; Koletsky *et al.*; Shubik). Tessmer and Brown reported a malignant skin lesion on the back of a bovine which had been exposed to fallout from an atom bomb test 15 years previously. Though malignancy usually develops at the site of chronic radiation dermatitis, as a result of repeated exposures to radiation, it may develop as a sequel to mild exposures with little chronic changes in the skin. It has been reported to occur in animals following a single exposure to beta radiation with little or no chronic change in the skin (Raper *et al.*).

In view of the superficial nature of most of the Marshallese lesions and the low evidence of chronic effects in the skin, the likelihood of skin cancer in this group seems diminished.

12.6.3 *Histopathology of Beta Lesions.* By and large, the histopathological

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FIG. 10. Development of pigmented nevus-like lesions in the neck region at the site of former beta burns in a 46-year-old woman (Case No. 78).

changes in the skin produced by beta radiation are much the same as those produced by gamma or x-rays. Since histological changes induced by the latter radiations have been well documented (MacKee, Cipollaro and Montgomery; Warren; Bloom and Bloom; and Walbach, etc.), a detailed description of the changes induced by beta radiation will not be presented. A limited number of studies of the histological changes in the skin of animals (Snider and Raper; Moritz and Henriques) and in man (Low-Beer) from beta radiation have been reported.

In general the changes produced by beta radiation are more superficial than those produced by more penetrating radiations with relatively much greater damage to the epidermis than to the dermis. With fallout radiation the damage is spotty in character with areas of damage surrounded by relatively normal tissue.

The histopathological changes induced in the skin by fallout in the Marshallese lesions were studied in sections of a number of biopsies taken during the first 7 weeks, at 6 months and at 2 years. Details of these changes can be found elsewhere (Conard *et al.*). Some of the major changes seen are summarized below. During the early, acute period of the lesions, the epidermis showed marked damage characterized by atrophy and flattening of the rete pegs with disorganization of malpighian and basal layers and marked cellular changes (pleomorphic nuclei, pyknosis and cytoplasmic halos). Additional features were atrophy or absence of the stratum granulosum, imperfect keratinization, and loose fibrillation and hyperkeratosis of the stratum corneum. Cells laden with pigment were frequently present

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throughout the epidermis. In the dermis the changes were largely confined to the upper part with edema, telangiectasis of vessels with perivascular infiltration of lymphocytes. Chromatophores filled with melanin were prominent. Figure 11 shows some of these changes in a pigmented lesion biopsied 3 weeks after exposure.

By 6 months there was considerable improvement in the histological appearance of the lesions. The following changes were found to persist in varying degrees: focal atrophy of the stratum granulosum, slight focal pigmentary disturbances in cells of the basal layer, and slight distrubances in polarity of the epithelial cells in basal papillary projections. In the dermis, telangiectasis of slight to moderate degree persisted.

At 2 years, biopsies at sites of persistent gross abnormalities revealed that none of the lesions were neoplastic or showed alterations suggestive of a precancerous condition. In some sections, acanthosis, absence of pigment in the basal layer and atrophy and benign dyskeratosis were noted in the malpighian layer of the epidermis. In the dermis degenerative changes in the collagen were noted frequently, and capillary dilation persisted.

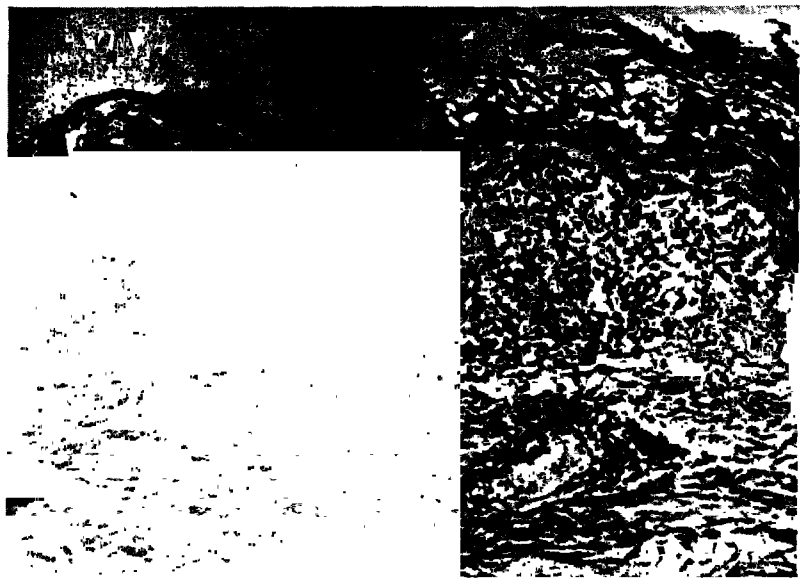

FIG. 11. Section from beta lesion of neck at 3 weeks after exposure to fallout. (× 100). Epidermis: extensive transepidermal damage (with slightly less involved zones on either side). Loose lamination of stratum corneum, absence of stratum granulosum. Parakeratinization with exfoliation of pigment containing cells. Disorganization of the malpighian layer. Dermis: mild edema of pars papillaris with indistinct capillary loops. Perivascular cellular infiltrate (lymphocytes and mononuclear phogocytes), in superficial corium with telangiectasis. Case 26.

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FIG. 12. Section (333 ×) from lesion on back of neck at 2 years after exposure to fallout. Lesion showed mottled pigmentation and depigmentation grossly. Section shows some loss of pigment in the basal layers of the epidermis and telangiectasis in the dermis. Case 39.

Some of these features may be seen in figure 12, which is a section taken at 2 years of a lesion on the back of the neck which showed gross pigment changes.

12.7 Therapy of Beta Lesions

The treatment of beta lesions during the acute stage is very similar to the treatment of thermal burns. Mild lesions will only require daily cleansing and application of bland antipruritic lotions and ointments. Calamine lotion with 1 per cent phenol is soothing. Analgesic and anesthetic ointments are helpful in allaying more painful symptoms and in keeping the skin soft in lesions that are dry and thickened. Antibiotics applied locally

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and/or parenterally should be used if secondary infection occurs, or prophylactically if the lesion is associated with severe leukopenia from whole body radiation. The above treatment proved quite adequate with the Marshallese lesions.

In severe lesions with the development of necrotic tissue, surgical debridement should be carried out. Use of pressure dressings, splinting and elevation of affected parts may be necessary. Early skin grafting should be considered in cases developing painful or progressive chronic radiation dermatitis (Brown *et al.*). For more detailed therapy of radiation lesions, the reader is referred to standard textbooks on the subject such as that of MacKee, Cipollaro, and Montgomery.

Several agents have been reported in recent years to be beneficial in the treatment of radiation lesions of the skin. Among these are preparations of the Aloe Vera plant (Lushbough; MacKee, Cipollaro, and Montgomery). The use of vitamins such as A and D are advocated by some investigators in the acute stages. Papomon *et al.* claim that use of fibrin films in combination with penicillin are helpful in treating radiation burns of the skin. Persing *et al.* have found Desitin ointment to be beneficial in the healing of beta burns in pigs. Nickson *et al.* have reported beneficial effects of parenteral triiodothyronine in reducing late effects of beta irradiated skin. Prenisone injections have been reported to reduce postirradiation inflammation (Matthewson). Further clinical experience with these agents is necessary before they can be recommended for general use in the treatment of beta burns.

A limited number of studies have been carried out to evaluate certain protective drugs and procedures in beta irradiated skin of animals. Davis *et al.* have demonstrated that pretreatment of rats with cysteine, sodium nitrite, and hypoxia protected rat skin significantly against ulcerating doses of beta radiation. However, they reported that postirradiation treatment with spleen homogenates gave less protection (believed to be nonspecific) and bone marrow homogenates did not affect the course of the injury. Chase has also demonstrated the effects of oxygen tension on radiation changes in the mouse skin in high altitude studies. Liebner *et al.* found that lowering the skin temperature of patients to 50° to 55° F. locally during irradiation had a "sparing" effect on the skin.

12.8 Factors Influencing Severity of Skin Lesions from Fallout

12.8.1 *Physical Factors.* Usually fallout material must be in contact with the bare skin to result in significant skin damage. Most of the lesions in the Marshallese occurred on exposed parts of the body, and protection was afforded by clothing, even a single layer of cotton material. Since clothing would probably not result in more than about 25 per cent attenuation of

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the beta particles, additional protection must have been afforded by the fact that the loosely-fitted clothing tended to hold the radioactive material away from the skin. Avoidance of skin contamination by taking shelter offers almost complete protection. No lesions developed in those Marshallese who remained in their houses during the fallout.

The ultimate dose to the skin depends on the radiation characteristics of the fallout material, the time after detonation that the fallout occurs, and the length of time that the material is in contact with the skin before decontamination is accomplished. Due to the process of radioactive decay which is quite fast during the first few hours, the earlier the time of the fallout, the greater is the dose rate from a given sample. This fact emphasizes the importance of early decontamination of the skin, particularly if contamination takes place during the first day after detonation. The fact that thorough decontamination of the Marshallese was not accomplished until their evacuation some 2 days after the accident, resulted in an appreciable increase of their skin dose. Those individuals that bathed or went swimming during the early period developed few lesions.

12.8.2 *Biological Factors.* There are certain *biological factors* known to influence the sensitivity of the skin to radiation. In addition to species differences referred to, it is known that the skin of certain parts of the body is more sensitive to radiation than that of others. In general, the thinner-skinned flexor surfaces of the body are more sensitive than the thicker-skinned extensor surfaces (MacKee, Cipollaro, and Montgomery). This was found to be true in the Marshallese. Lesions were more prevalent on the front and sides of the neck, axilla and antecubital fossae. Another factor is associated with pigmentation of the skin. Darker-skinned people, brunettes, are known to be less sensitive to radiation than blondes or people with ruddy complexions, and Negro skin is the most resistant (MacKee, Cipollaro, and Montgomery; Bloom and Bloom).

Areas of the body where perspiration is more profuse, such as the folds of the neck, axillae, and antecubital fossae tend to cause the fallout to stick and collect. It was found that skin lesions in these areas were more abundant in the Marshallese. This effect is increased in a warm, humid climate, such as in the Marshall Islands.

12.9 Concluding Remarks

As a result of the Marshallese accident, the potentialities of serious injury to the skin from fallout associated with the detonation of large nuclear devices are apparent. Of concern also is the occurrence of similar radiation injuries to the skin from accidental exposure to radioisotopes which are being used increasingly.

The skin hazards associated with fallout can be greatly reduced by taking

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simple precautionary measures. Much was learned from the Marshallese experience in this regard. This group of people was not aware of the hazards of fallout and only minimal, if any, efforts were made to protect themselves. This situation represents an extreme example, and the extensiveness of the skin effects could have been greatly reduced had proper measures been taken. Based on the experiences of these people during the critical fallout period and the skin lesions that developed on an individual basis, the following facts emerge:

1. Avoidance of contact of fallout material on the skin by taking shelter or covering the body with clothing virtually eliminates the possibility of skin effects.

2. Prompt, thorough decontamination of the skin and hair is of utmost importance. Repeated scrubbing with soap or detergent and water may be necessary. If contamination of the hair is severe, it may be advisable to clip the hair close or shave the head.

3. Areas of the body where perspiration is more profuse tend to cause the fallout material to collect. Such areas should be carefully checked for contamination. A warm, humid climate will naturally aggravate this effect.

4. Moderately severe beta lesions of the skin and epilation may result from fallout situations in which the whole body penetrating dose of radiation is sublethal. With such doses, the skin lesions do not appear to complicate the radiation syndrome.

5. In situations where skin lesions are associated with larger doses of whole body radiation with marked leukopenia, such lesions might become secondarily infected more easily and afford portals of entry leading to bacteremia or septicemia.

6. Severe skin irradiation with minimal whole body irradiation might result in fallout situations where prompt evacuation from the contaminated area occurred, but skin decontamination was delayed.

7. Early skin and eye symptoms might be mildly disabling during the first day or two after exposure to fallout and later symptoms associated with full-blown lesions might be quite disabling. Late effects on the skin in the form of chronic radiation dermatitis and malignancy are possible complications.

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